Journal of
Cardiology & Cardiovascular
Surgery

Case Report

DOI: 10.51271/JCCVS-0028

Spontaneous recovery in the early period of cardiac remodeling due to carbon monoxide poisoning

Bilge Tanık[1], Ahmet Bilal Kandemir[2], Sevgi Örs[2], Yücel Yılmaz[1]

[1]Department of Cardiology, Kayseri City Hospital, Kayseri, Turkiye
[2]Department of Anesthesiology and Reanimation, Kayseri City Hospital, Kayseri, Turkiye

Cite this article: Tanık B, Kandemir AB, Örs S, Yılmaz Y. Spontaneous recovery in the early period of cardiac remodeling due to carbon monoxide poisoning. *J Cardiol Cardiovasc Surg.* 2024;2(1):21-23.

Corresponding Author: Bilge Tanık, drebilgetanik@gmail.com

Received: 04/03/2024 ◆ **Accepted**: 19/03/2024 ◆ **Published**: 29/03/2024

ABSTRACT

Carbon monoxide can cause cardiac injuries, including transient left ventricular dysfunction, by causing hypoxia at the tissue level. In this case, we present a case that developed serious global left ventricular dysfunction (ventricular remodeling) as a result of carbon monoxide poisoning and recovered very early. Although we could not use any cardioprotective agents in the treatment of the patient without hyperbaric oxygen, we experienced that the left ventricular functions completely recovered on the 7th day of hospitalization.

Keywords: Carbon monoxide poisoning, acute left ventricular dysfunction, hypoxia

INTRODUCTION

Carbon monoxide (CO) occurs as a result of carbon-based fuels not burning properly. It can cause acute and chronic poisoning in humans (alives).[1,2] CO's affinity for hemoglobin (Hb) is 220 times higher than oxygen. By binding to Hb, CO reduces the oxygen-carrying capacity of the molecule (it both competes with oxygen and causes structural changes in Hb).[3,4] Exposure to high concentrations of CO can have fatal consequences and is one of the most common causes of death due to poisoning worldwide. CO can cause cardiac injuries by causing hypoxia at the tissue level.[5] Cardiac symptoms such as myocardial ischemia, heart failure, and arrhythmia have been reported after exposure to CO.

While 4 of the 45.000/500 million people affected by COP die every year in the USA, but these data are not clear in our country.[6] COP can affect all systems, including the heart, brain and nervous system, muscle, gastrointestinal system, and skin. Since neurological and cardiac functions are affected in the early period, the first symptoms are usually seen in the neurological and cardiovascular systems.[7] Clinical severity depends on the amount of CO inhaled, its duration, and the current health status. There is no specific antidote. The most commonly applied treatment methods are normobaric and hyperbaric oxygen therapy.[8]

In this article, we will present a case of severe left ventricular dysfunction in a young female patient, which developed in the early period after COP and resolved spontaneously.

CASE

A 29-year-old woman was found unconscious in her patient room and was taken to an emergency room at another medical center.

The patient, whose Glasgow coma scale (GCS) was evaluated and found to be low, was intubated (GCS: 3). Since the room she was staying in was heated with a stove and the CO levels in her blood were between 20% and 30%, she was diagnosed with COP and sent to our hospital for treatment and admitted to the general intensive care unit.

In the history taken from the patient's relatives, it was stated that he had no known chronic disease and did not use cigarettes, alcohol or drugs. During the cardiovascular system examination, blood pressure arterial measurement was evaluated as 70/56 mmHg and pulse rate was 110/min. S1 and S2 heart sounds were natural, and no murmurs were heard. In the respiratory system examination, respiratory rate was evaluated as 30/min and blood oxygen saturation was evaluated as 98% with ventilator support. Lung sounds were natural bilaterally and both hemithoraxes participated equally in breathing. No pathological appearance was observed on electrocardiography other than sinus tachycardia (Table 1) (Figure 1). No obvious pathology was seen on the posterior-anterior chest radiography when the intubation tube was broken. Abdominal examination was normal. The laboratory results of the intensive care unit admission were aspartate aminotransferase (AST)/alanine aminotransferase (ALT):45/33 creatine kinase (CK):3113, creatine kinase isoenzyme MB (CK-MB):85, aminoterminal part of B-type natriuretic peptides (NT-Pro BNP):1887 and changes in these patient outcomes throughout treatment are summarized in Table 2. Laboratory results other than these were evaluated to be within the normal range. General supportive treatment (fluid support, daily electrolyte replacements, antiemetic treatment, respiratory support, etc.) and hyperbaric oxygen therapy were planned for the patient. Cardiology consultation

This work is licensed under a Creative Commons Attribution 4.0 International License.

was requested from the patient because he was hypotensive on the day of admission and his cardiac troponin I values were high. To evaluate the level of cardiac involvement, transthoracic echocardiography was performed with a device with a 3.5 MHz transducer (Vivid 5 GE Medical System, Horten, Norway)). In echocardiography, LV ejection fraction was evaluated as 30-35% and global hypokinesia was detected in left ventricular wall movements (Figure 2). No pathological findings were detected in other echocardiographic measurements and valve evaluations. Since the patient's general condition was not good and inotropic support continued, classical LV remodeling and/or LV insufficiency drug treatments such as B-blockers, angiotensin-converting enzyme inhibitors, etc. could not be given. The patient was given hyperbaric oxygen therapy for 4 days. In the control echocardiography performed on the seventh day of the patient's hospitalization, it was observed that the LVEF improved (55%) (Table 1). The patient, who had no additional problems, was discharged from the general intensive care unit on the 15th day without any cardiac medical treatment planned. Service monitoring was continuing at the time this article was written.

Table 1. Echocardiographic and electrocardiographic results of the patient throughout the treatment process

Days	Echocardiography		Electrocardiography
	(Ejection fraction %, Simpson metod)	Wall motion	
1	30-35	LV global hypokinetic	Sinus tachycardia
3	35	LV global hypokinetic	Sinus tachycardia
5	40-45	All walls slightly hypokinetic	Sinus rhythm
7	55	-	Sinus rhythm

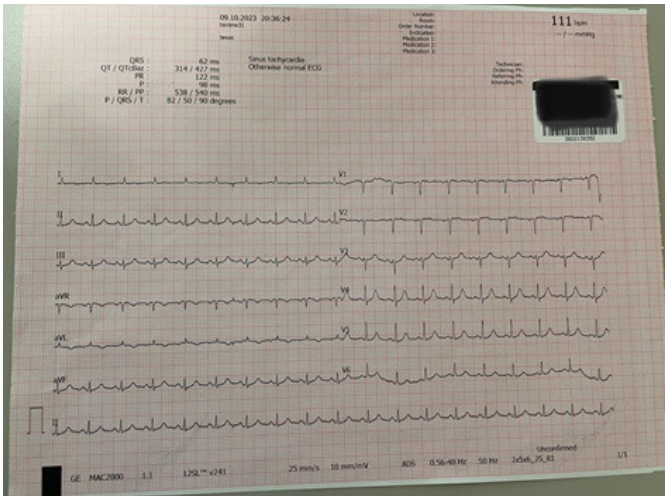

Figure 1. Electrocardiography performed when she was admitted to the general intensive care unit

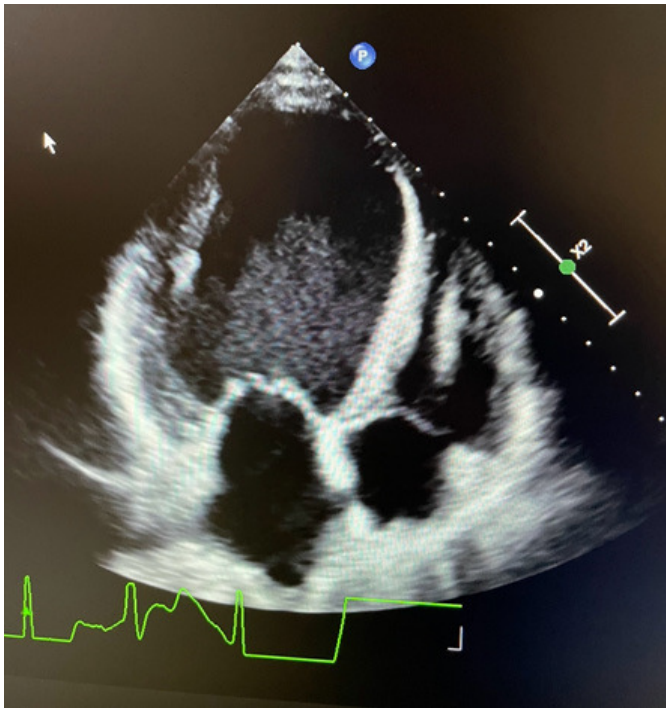

Figure 2. Echocardiography performed when she was admitted to the general intensive care unit

DISCUSSION

In this article, a case of left ventricular remodeling that occurs in the early period after COP and resolves spontaneously is presented. In our case, the recovery time for left ventricular remodeling was 7 days.

In COP, hypoxia due to inadequate oxygen delivery is the cause of the basic symptoms and signs. In most cases, cardiovascular findings may be missed or diagnosed late due to respiratory and neurological symptoms that predominate.[9] While myocardial damage and fibrosis may be observed at low dose exposures, it has been previously reported in the literature that fatal arrhythmias may occur at high dose exposures. In addition to hypoxia due to COP cardiotoxicity, CO gas also has a direct toxic effect by inhibiting cytochrome oxidase on myocyte mitochondria.[10]

In 1865, CO-induced heart failure and myocardial ischemia were described for the first time.[11,12] In later years, various cardiac clinical diseases such as CO-induced arrhythmias, electrocardiographic changes, acute myocardial infarction, pulmonary edema, and cardiogenic shock were reported.[11,13,14] Those with a previous history of cardiovascular disease are more sensitive to CO-induced cardiotoxicity.[15] Tachycardia is the most common cardiovascular finding.[16] Chest pain occurs regardless of the presence of coronary artery disease due to myocardial ischemia or necrosis.[16,17] Shortness of

Table 2. The laboratory results of the patient throughout the treatment process

Days	Tro I (ng/ml)	CK (U/L)	CK-MB (U/L)	AST/ALT (U/L)	Cre (mg/dL)	GFR (ml/dk)	Sodium (mEq/L)	Potassium (mEq/L)	Calcium(mg/dL)
1	541	3113	85	45/33	0.66	120	140	4.5	7.4
3	423	2809	59	675/1183	0.92	84	151	4.1	7.2
5	230	2222	37	170/997	0.64	121	150	3.7	7.1
7	164	473	22	58/303	0.55	127	137	3.7	7.7

Abbreviations; Tro I; Troponin I, CK; Creatine kinase, CK-MB; creatine kinase isoenzyme MB, AST/ALT; Aspartate aminotransferase/alanine aminotransferase, Cre; Creatine, GFR; Glomerular filtration rate

breath may be due to hypoxia or to depression in heart function.[18] Shortness of breath and tachycardia were detected in this patient at the time of admission, and these symptoms suggested the presence of cardiac dysfunction.

Diffuse or segmental wall motion abnormalities may occur in patients exposed to CO. Echocardiography may be considered, especially in cases of unexplained hypotension, myocardial damage detected by laboratory tests, and in patients with shortness of breath without lung pathology. Satran et al.[19] showed in a study that the left ventricle was affected by 57% after CO exposure. Additionally, it was determined that the decrease in LV systolic functions was proportional to the amount of CO exposure. Park et al.[20] found dysfunction in the LV in 29% of the patients in their study and reported global involvement in only 5% of the patients. Information about the recovery period of this depression, which is generally described as reversible, is very limited. Jang et al.[5] reported in their case report that the general condition was better and that severe left ventricular systolic dysfunction improved on the 4th day by receiving heart failure treatment. Lee et al.[21] reported that 18 of 21 people who had follow-up echocardiography recovered within 3 days by receiving heart failure treatment. However, in this series, both the patients' ages were older and their LVEF was around 53% on average. Although our case is similar to the cases of Jang et al.[5], in our case the general condition of the patient was much worse (probably the CO exposure time was longer) and although he could not take heart failure medications due to hemodynamic compromise, we still detected an improvement in a short time.

This study gives an idea that left ventricular dysfunction after COP can improve in a very short time, there is no need to pass for a long time for control echocardiography, and perhaps there may be no rush to start heart failure treatment. However, case-controlled clinical studies are needed for more definitive results.

CONCLUSION

This study suggests that left ventricular dysfunction after COP can improve quickly, there is no need to wait a long time for control echocardiography, and there may be no rush to begin heart failure treatment. More definitive results, however, will require case-controlled clinical studies.

ETHICAL DECLARATIONS

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. State of the art. Health effects of outdoor air pollution. *Am J Respir Crit Care Med*. 1996;153:477-498.
2. Kandiş H, Katırcı Y, Karapolat BS. Karbonmonoksit zehirlenmesi. *Düzce Med J*. 2009;11(3):54-60.
3. Kandis H, Katırcı Y, Çakır Z, Aslan Ş, Uzkeser M, Bilir Ö. Acil servise karbonmonoksit entoksikasyonu ile başvuran olguların geriye dönük analizi. *Akad Acil Tıp Derg*. 2007;5(3):21-25.
4. Piantadosi CA. Carbon monoxide poisoning. *Undersea Hyperb Med*. 2004;31:167-177.
5. Jang W, Park JH. Transient left ventricular systolic dysfunction associated with carbon monoxide toxicity. *J Cardiovasc Ultrasound*. 2010;18(1):12-15.
6. Weaver LK. Carbon monoxide poisoning. *N Engl J Med*. 2009;360(12): 1217-1225.
7. Brown SD, Piantadosi CA. In Vivo binding of carbon monoxide to cytochrome c oxidase in rat brain. *J Appl Physiol*. 1990;68(2):604-610.
8. Jain KK. Textbook of Hyperbaric Medicine. In: Jain KK, ed. Carbon monoxide and other tissue poisons. 6th ed. Springer International Publishing: 2016:131-154.
9. Fiorista F, Casazza F, Comolatti G. Silent myocardial infarction caused by acute carbon monoxide poisoning. *G Ital Cardiol*. 1993;23(6):583-537.
10. Peers C, Steele DS. Carbonmonoxide: a vital signalling molecule and potent toxin in the myocardium. *J Mol Cell Cardiol*. 2012;52(2):359-365.
11. Gandini C, Castoldi AF, Candura SM, et al. Carbon monoxide cardiotoxicity. *J Toxicol Clin Toxicol*. 2001;39(1):35-44.
12. Marius-Nunez AL. Myocardial infarction with normal coronary arteries after acute exposure to carbon monoxide. *Chest*. 1990;97(2):491-494.
13. Anderson RF, Allensworth DC, DeGroot WJ. Myocardial toxicity from carbon monoxide poisoning. *Ann Intern Med*. 1967;67(6):1172-1182.
14. McMeekin JD, Finegan BA. Reversible myocardial dysfunction following carbon monoxide poisoning. *Can J Cardiol*. 1987;3(3):118-121.
15. Allred EN, Bleecker ER, Chaitman BR, et al. Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. *N Engl J Med*. 1989;321(21):1426-1432.
16. Thompson N, Henry JA. Carbon monoxide poisoning: poisons unit experience over five years. *Hum Toxicol*. 1983;2(2):335-238.
17. Ebisuno S, Yasuno M, Yamada Y, et al. Myocardial infarction after acute carbon monoxide poisoning: case report. *Angiology*. 1986;37(8):621-624.
18. Diltoer MW, Colle IO, Hubloue I, et al. Reversible cardiac failure in an adolescent after prolonged exposure to carbon monoxide. *Eur J Emerg Med*. 1995;2(4):231-235.
19. Satran D, Henry CR, Adkinson C, Nicholson CI, Bracha Y, Henry TD. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. *J Am Coll Cardiol*. 2005;45(9):1513-1516.
20. Park JS, Seo KW, Choi BJ, et al. Various echocardiographic patterns of left ventricular systolic dysfunction induced by carbon monoxide intoxication. *Cardiovasc Toxicol*. 2016;16(4):361-369.
21. Lee JH, Kim HS, Park JH, et al. Incidence and Clinical course of left ventricular systolic dysfunction in patients with carbon monoxide poisoning. *Korean Circ J*. 2016;46(5):665-671.